patient surviving the operation for over two weeks. During that time inflammatory changes and invasion of the malignant growth into the brain very much obscured and complicated the anatomo-pathologic conditions giving rise to the symptoms during life. It would, above all, have been of importance to ascertain to what degree and in what manner the cortex was changed. The topography of these changes might possibly have thrown more light on the question of a trophic cerebral centre.

INFECTIOUS ENDOCARDITIS, WITH SEPTICÆMIA, COMPLICATED WITH MULTIPLE NEURITIS.

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AND

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The recognition of various septic or infective agents as causes of multiple neuritis is, of course, not entirely new. The mind recurs at once to such familiar instances as diphtheritic paralysis and to the less frequent but well-known examples of neuritis which occur after typhoid fever, smallpox, and other infectious diseases. More recently still the rather rare peripheral palsies seen after childbirth have been, perhaps justly, attributed to sepsis rather than to pressure. But this term "sepsis" has still a very wide and indefinite application, and seems to be used to include a large number of disease-processes—probably dependent upon a variety of pathogenic organisms—which have not as yet been differentiated and labelled. The infection following childbirth is probably not always the same in all cases, nor, again, is it probably always the same as that following upon a poisoned wound or a suppurating ear. Whatever their source or their exact nature, it has long been recognized that some peculiarly active and malignant disease-agents fasten upon the endocardium and upon the lining membrane of the aorta, whence, as from a centre, they proceed to sow broadcast their baleful effects.

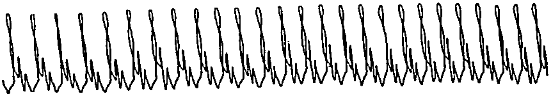
It is not, indeed, necessary to assume that these disease-agents are always the same. We know, on the contrary, that specific and distinct infective agents attack the endocardium with varying degrees of activity, as, for instance, those of rheumatism, of chorea, and of scarlet fever. But these diseases have other and distinctive clinical features which serve to mark them. There are, however, some, perhaps not a few, active and as yet unnamed, but very malignant forms of infection, making their habitat especially in the heart and great bloodvessels, which, because of their wide systemic effects, and because of their occasional disastrous consequences upon the nervous system, merit our special attention here. The commonly-described and rapidly-fatal ulcerative endocarditis is not the only form whose infection is to be feared, because there may be a non-ulcerative vegetative form, somewhat slower in its action, but none the less eventually fatal. This is marked by immense masses of vegetations, by heart symptoms sometimes obscure but sometimes conspicuous, by a true hectic or typhoid type of fever, by sweating and emaciation, and by various involvements of the nervous system, among the least common but most interesting of which may be neuritis. Multiple abscesses of the brain may also be observed. As an example of each of these we have a case to report here and specimens to present.

E. O., a middle-aged male; had been a steady drinker for years. He denied syphilis. He had been much exposed to hard out-door work, and during the past severe winter had earned a living by shovelling snow. In January, while thus employed, he was taken rather suddenly ill. He had fever and severe pains in his neck, back, shoulders, and limbs. He did not keep constantly in bed at first, but he continued to grow feebler, until one night he fell because of his weakness, and thereafter was obliged to remain in bed. He continued with fever and rapid emaciation for almost three months, losing over forty pounds during this period before his admission to the hospital. From the statements of the patient and his friends, and from his appearance when admitted, it seems probable that during this period of three months he had had a persistent hectic fever, with progressive emaciation and paralysis.

On admission to the hospital, early in April, the patient presented a rather anomalous appearance. He had two sets of symptoms which were prominent and clearly demarcated from each other. The first of these was a low typhoid type of fever, with rapid pulse and malnutrition; the second was an irregularly-distributed multiple neuritis. He was a dark-complexioned, much emaciated man, with a skin of peculiar swarthy or dirty yellowish-brown hue. The face had an expression of hebetude. The tongue was dry and coated, and had a bright red tip. The man's appearance suggested long neglect.

The typhoid type of symptoms was marked by a continued febrile course of temperature. On his admission the temperature was 101⅗°, and during his entire sojourn in the hospital, *i. e.*, for five weeks, the bodily heat was above normal. If to this period are added the three months before admission, during which he had evidently had the same wasting fever, we have a febrile course of more than four months. At first rather hectic, it became much less remittent toward the end. The pulse was weak and rapid, as high sometimes as 120, but occasionally falling to almost normal. This pulse and the heart-symptoms at once attracted attention, and eventually led to the correct diagnosis. The heart, for instance, had a booming first sound, with marked accentuation of the second pulmonic sound. A loud aortic diastolic murmur was heard, best in the third interspace at the *left* of the sternum. The apex beat was diffuse, heaving, and displaced downward to the left. The pulse was of the so-called "water-hammer" or "Corrigan" type; and this persisted almost until the end. It was unique. The artery bounded against the finger with much force; the blood, as it were, pausing for an instant as if in an eddy, then suddenly sinking away from under the touch. The accompanying sphygmogram shows this quickness of the pulse, the tracing of the downstroke coinciding almost with that of the upstroke. The loop is caused by the violent oscillations of the artery. The secondary waves are well marked (Fig. 1). Tracings could also be made from the ulnar artery, the pulsation of which was almost as strong as that of the radial artery.

FIG. 1.



The patient, as said before, resembled superficially one suffering from typhoid fever. He had the mental dulness, the wandering delirium, the dry-coated tongue, the rapid pulse, the continued fever, and the general asthenia and wasting. On the other hand, he had a good appetite, no bronchitis, no eruption, no typhoid stools, and the belly was scaphoid. Nor did the range of temperature, although constantly high, correspond strictly to that of the temperature of typhoid fever; there was not the regularity of evening rise and morning decline. Hence, the diagnosis of enteric fever was rejected.

But, as already said, the patient had a second group of symptoms quite as striking as those that characterized his typhoid state. He had very plainly the symptoms of a multiple neuritis. This was irregularly distributed.

The motor phenomena consisted in complete paralysis of the left arm, partial paralysis of the right arm, and double wrist-drop, more pronounced on the left side. The right hand had a tendency to assume the position of *main-en-griffe.* The legs could be moved, but power and nutrition in them were impaired. The knee-jerks were absent. There was no foot-drop. To the faradic current there was diminution in response in the left arm; no response at all in the extensors of the forearm, except in the extensor carpi ulnaris. The muscles of the right arm responded more actively than did those of the left. In the legs the response to faradism was sluggish; in the thighs it was good.

(The galvanic tests were not made, because of some defect in the battery. Later, when the battery had been repaired, the patient was so weak that it was thought best not to transport him to the battery-room, especially as the faradic tests had been quite conclusive.)

The feet and legs were slightly œdematous; the left hand and forearm, also, were somewhat swollen.

Marked wasting of the muscles of the shoulder girdle and of the arms, forearms, and hands was present. This was more noticeable on the left side. There was not marked wasting of the muscles of the legs. No fibrillary twitching, nor any spastic or increased myotonic state of any of the muscles was observed.

Sensory symptoms were not conspicuous. There was no distinct anæsthesia, but merely some retardation of sensation. No thermo-anæsthesia.

After admission the condition of the patient presented no striking change, only a progressive decline. He continued to grow more paralyzed, and the muscles of the arms and hands wasted more and more. Withal he had no pain, but he had a peculiar rigidity of the neck, which made turning somewhat painful to him.

The diagnosis of the disease was not easy at first, because the grouping of the two sets of symptoms made the man's condition enigmatical. Typhoid fever, which the malady resembled most at first glance, was excluded, as already said, by the range of temperature, the absence of the rose-colored spots, the scaphoid belly, and the absence of the characteristic stools. That there was some septic or infectious process seemed certain, because of the persistent hectic temperature, the sweating, the delirium, the involvement of the heart and pulse, and the albuminous urine. That the man had multiple neuritis was evident, and he had the history of exposure and alcoholism to explain it, in part at least; but the diagnosis of multiple neuritis of alcoholic origin could not be made conveniently to include his typhoid conditon, which had existed now for many weeks. In our experience simple alcoholic multiple neuritis is not accompanied with a hectic state of many weeks' duration. The suggestion of a septic or ulcerative, endocarditis occurred to our minds as the most plausible explanation of the symptoms; and this diagnosis was established eventually by the discovery of a persistent and gradually increasing regurgitant aortic murmur, with its accompanying "water-hammer" pulse. I say "established," because while this diagnosis is regarded usually as problematical to a certain extent, it was not so in our case. The lesion seemed to assert itself so very conspicuously under our observation as the case progressed that, with its attendant hectic phenomena, it left no reasonable doubt of its accuracy. The association of a multiple neuritis with this systemic infection was the phenomenon which to our eyes gave the case a unique and paramount importance, and to which reference will be made again. In the expectation of possible embolic processes in the spleen and kidneys, the splenic region was examined repeatedly for tenderness and the urine for blood corpuscles. Neither were observed, although the autopsy, as will be seen later, showed infarcts in the spleen and in one kidney.

About three weeks after admission our diagnosis of a septic endocarditis was much strengthened by the appearance of small purpuric spots scattered over the body. This eruption was characteristic of a process of blood-poisoning. The patient continued to have a rather high rise of temperature at irregular intervals, and had frequent profuse, drenching sweats. A few days later a bedsore developed over the sacrum. Sudamina also appeared.

The patient's strength gradually failed. Stupor, carphologia, feeble pulse, and high temperature marked the closing days. He died five weeks after admission to the hospital.

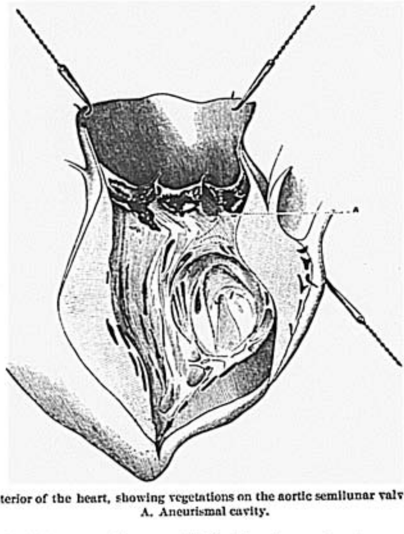
Notes of autopsy. Body of a much emaciated, middle-aged man. The muscles of the chest and arms are wasted. In each hand the atrophy of the abductor indicis is very marked. The fingers are flexed at the first interphalangeal joint. The scapular muscles, especially the supraspinatus and lower portion of the trapezius, are much wasted. The buttocks and hips are the seats of extensive decubitus.

Abdominal cavity. With exceptions to be noted, the contents are normal. No lesions of typhoid fever in the intestines.

Thoracic cavity. The pleuræ are bound together on each side by adhesions of moderate firmness. The lungs are rather œdematous, but otherwise normal. The pericardium contains an excess of fluid, there being about two and a half ounces of yellow, translucent serum. The parietal pericardium shows a milk spot.

Heart. Enlarged; left ventricle firm. Right ventricle contains a small quantity of fluid blood and a chicken-fat clot which has long ramifications through the auricle into the veins. The tricuspid orifice is large and easily admits four fingers and the thumb. Mitral orifice is normal in size, admitting two fingers. Left chambers almost empty. Left ventricle contracted. A small chicken-fat clot is in the left auricle and aorta; also in the pulmonary artery. The right ventricle seems to be dilated; the wall 6 mm. thick; the wall consists almost entirely of columnæ carneæ. The tricuspid valve is thickened along its edges. The pulmonary valves are normal, except for some thickening along the free

FIG. 2.



Interior of the heart, showing vegetations on the aortic semilunar valves.
A. Aneurismal cavity.

margins of one of the leaflets. Mitral valve is thickened, with a tendency to atheromatous change in the anterior leaflet. The chordæ tendineæ of the anterior leaflet are unusually thick. The heart-muscle is lighter in color than normal and diminished in firmness, evidently on account of degeneration. The papillary muscles especially are light in color.

The *aortic valves* present an astonishing condition. Springing from the inner surface of each leaflet there are huge, cauliflower-like vegetations, which are soft, compressible, slightly injected, the protuberances here and there spanned by threads of lymph. (Fig. 2.) The excrescences are longest and thickest on the anterior and middle leaflets. The posterior leaflet is covered by a large number of vegetations, but of small dimensions. The growths spring from the valve a little below the free margin. On the anterior leaflet the base of the growth extends clear across the valve; on the middle leaflet, two-thirds of the way across. On the posterior the base of the vegetation covers a little more than half of the leaflet. The length of the anterior growth is 2 cm., of the middle also 2 cm. The thickness is 6 mm. in the first and 3 mm. in the middle growth. Vegetations also spring from the angular space between the valves. At the junction of the anterior and posterior leaflets there is a deep cavity, which is surrounded by vegetations, has a roughened interior, and looks almost like a small aneurism. (Fig. 2, A.) The outer surfaces of the valves (facing the aorta) are ridged, and in the position of the vegetations show a funnel-shaped depression running into the vegetations. The intima of the aorta is intensely atheromatous, showing sclerotic and calcareous patches. Weight of heart 14 ounces.

The *spleen* is slightly enlarged. Patches of old perisplenitis are seen, and in the upper part there is a characteristic anæmic infarct. It is a large, white, wedge-shaped mass, extending from the cortex inward a distance of 2½ cm., and measuring 4½ cm. in width at its base. Weight of spleen 6½ ounces.

Suprarenals normal.

Kidneys. In the left kidney the renal arteries stand out prominently; the kidney is firm and normal in size; the capsule strips quite readily, leaving a smooth surface. Weight 5 ounces. In the right kidney the ureter is somewhat dilated; the kidney is normal in size; on its anterior surface are one large and one small yellow patch, which, on section, are seen to be the bases of wedge-shaped masses. In appearance these are white infarcts, like that in the spleen. The capsule strips readily. Weight 4½ ounces.

The *liver* weighs 57 ounces. On section it shows prominence of the interacinous connective tissue. The other abdominal organs are normal.

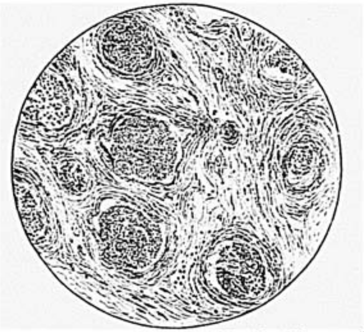
The *aorta* is very atheromatous, especially in the arch and thoracic portion, where there are numerous large calcareous plates.

Brain. The membranes are somewhat œdematous, the pia strips readily. The vessels of the circle of Willis are distinctly thickened; from the right middle cerebral artery, within the fissure of Sylvius, springs a small yellowish body a little larger than a grapeseed, quite firm; it is not a dilatation of the artery; it rests in the brain-substance like a foreign body; no area of inflammation or softening surrounds it; it may be a small infarct similar to that in the spleen. On removing the dura from the cord, thin whitish, scaly patches are seen on the pia about the middle of the cord; these scales are not calcareous, but of cartilaginous firmness. Weight of brain 45½ ounces. In the *cord* itself nothing pathological is discoverable with the naked eye.

Description of Sections.

Musculo-spiral nerve. Right. This nerve shows marked increase of the connective tissue between the fasciculi. (Fig. 3.) This increase is quite apparent even to the naked eye on holding the section up to the light. Hence this is not a pure parenchymatous neuritis. The individual fasciculi also show in some instances decrease in the number of nerve-fibres and increase in the connective tissue. Some fasciculi exhibit bundles of long, opaque fibres in places where the fasciculus has been cut in an oblique direction. In one place a much thickened bloodvessel is seen, and in several places obstructed vessels.

FIG. 3.



Section of the musculo-spiral nerve, showing proliferation of the connective tissue.

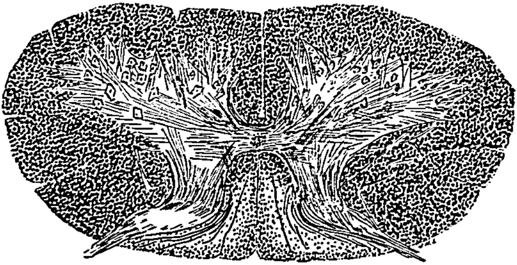
Left. About same appearances as in right.

Ulnar nerve. Right. A much thickened and obstructed bloodvessel is seen in one place near the periphery. There is also general increase of the connective tissue, but the individual fasciculi are in better state than in the musculo-spiral nerve. About some of the obstructed bloodvessels, and also about one of the fasciculi—here nearly empty—there is an accumulation of leucocytes. In some places in these sections proliferated connective tissue in concentric layers around bloodvessels is seen (as shown in Rosenheim's illustration as found in Dana's *Text-book*, p. 69). These are probably identical with the "islets of sclerosis" found by Gombault in his case of Landry's paralysis.

NOTE.—In all these sections the nerve fibres have stained heavily—Weigert's stain—and it is not easy to state exactly whether individual fibres are degenerated or not. The general appearance, however, of all these sections leads to the opinion that we have here a connective-tissue proliferation—*i. e.*, a true inflammation, which includes in some instances the fasciculi, but that the effects upon the fibres are chiefly secondary or degenerative. It is in accord with the ideas of modern pathology to expect to find the results of inflammatory processes in connective tissue and about bloodvessels, as we do in this case. The occurrence of a primary parenchymatous neuritis, as advocated by some, especially Gowers, if by this is meant a precedent and independent involvement of the axis-cylinder, is not so in accord with modern ideas; and this involvement must be an exceedingly difficult thing to demonstrate, even by the most expert pathologist. Certainly most of the illustrations given in the books do not convey such an idea.

Spinal cord. The interesting fact is noted that in the cord, otherwise healthy, there is some evidence of degeneration in the posterior columns. In the cervical region this is marked in both the columns of Goll and Burdach (Fig. 4). In the lumbar enlargement, however, the regions of the root-zones are most involved. This association of posterior sclerosis with multiple neuritis has been noted by others. The anterior horns in all the sections examined have a full supply of multipolar cells, and the lateral pyramidal tracts are in nowise affected.

FIG. 4.



Cervical enlargement of the spinal cord from a case of infectious endocarditis with multiple neuritis, showing degeneration at this level of both postero-median and postero-external columns, with preservation of small strip along postero-median septum.

It is well to remember, in examining these sections, that they have been made from the nerve-trunks at some distance from the periphery. As is well known, the most marked changes are usually in or near the peripheral nerve-endings. Hence, in this case the sections are from a portion of the nerves probably some distance above the areas of greatest change.

The association of a septic process and multiple neuritis was the phenomenon which gave our case an especial interest. The explanation of this association is, of course, the one point of scientific importance. Was the association a mere coincidence, or was the septic process the cause of the neuritis?

Several points must be considered in this connection. First, the septic process was probably antecedent to the neuritis, as shown by the history. The exhausting, wasting hectic fever had existed for many weeks before the patient's admission to the hospital. It was the most conspicuous of the two sets of symptoms, and the one that caused the fatal end. The patient died of sepsis, not of neuritis. The origin of this sepsis is, it is true, problematical. It was not in any way consequent upon a nerve-lesion, as is sometimes seen in cord-cases, in which a septic infection occurs from a bedsore. This patient had no bedsore nor any break in his skin when admitted to the hospital, far advanced in his disease, and no bedsore developed until toward the end. Again, it may be observed that this septic process did not depend upon a pus-forming bacillus. The vegetations within the heart and the infarcts in the spleen and the kidney were not attended with the formation of pus. An exception was possibly observed at the base of one of these masses, where what seemed to be a small ulcerated cavity was found. There were no multiple abscesses as in cases of pyæmia. In this respect this systemic poison resembles some of the other great nerve poisons, as, for instance, those of tetanus, hydrophobia, chorea, and diphtheria, in none of which is there a formation of pus. The origin of the infection in the heart in our case is obscure.

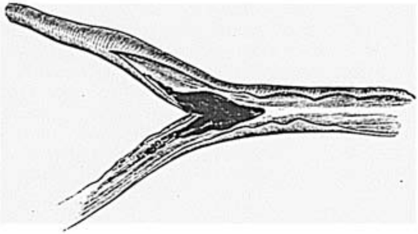
Finally, the neuritis was not so extensive and severe as occurs in some cases that recover. It was only irregularly distributed, and not, as in most fatal cases of neuritis due to alcohol, symmetrically and extensively spread. In fact, it does not seem probable that the neuritis, severe as it was, would have killed the patient without the septic process. On the other hand, there is evidence, no doubt, in favor of an alcoholic origin of the neuritis. The patient had been a steady drinker for years, and this excess was associated with hard labor and exposure—an association which, more than any other, precedes the onset of this disease.

A bacteriological examination of these cardiac vegetations would have been a most desirable conclusion to our study of this case, but was not possible at the time.

In conclusion, we would call attention to the extreme importance of these immense giant vegetations in the heart, and occasionally in the aorta, as causes of obscure but grave symptoms, both general and nervous. Only a few days ago, and since this report was made up, a man died in the hospital service of one of us with symptoms simulating closely those of typhoid fever. His case was briefly as follows:

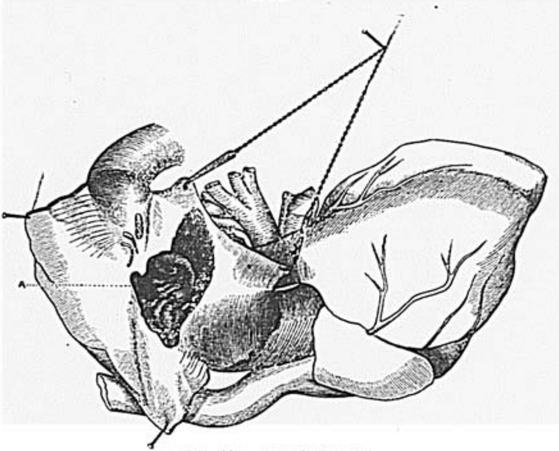
A. B., male, aged about forty-five years, was admitted to the hospital suffering with headache, hebetude, psychic disturbance, such as melancholia, and an almost persistent but variable fever. The headache was frontal, and apparently severe, for the patient, even in his hebetude, complained constantly of it. The melancholia was also a conspicuous symptom; it was associated with delusions of personal unworthiness. When roused and questioned he reverted constantly to the subjects of his melancholic delirium, and sometimes he wept about them. There

FIG. 5.



Brachial, radial, and ulnar arteries, with embolus at bifurcation

FIG. 6.



A. Polypoid vegetation in the aorta.

had been, also, some diarrhœa and abdominal distention, so that the case simulated rather closely typhoid fever. No rose-colored spots, however, could be found at any time, and the temperature, instead of ranging constantly above normal, dropped occasionally to 98° or below that point, as it is apt to do in septic cases. The diagnosis of the case remained quite obscure. There were no lung-symptoms, and nothing wrong was found in the urine except some albumin. There was a history of exposure to the sun, which suggested at first heat-stroke. Later, tuberculosis was suggested, searched for and excluded. No cardiac symptoms were present, as the mass found post mortem in the aorta did not embarrass the heart's action in the least. Absence of the left radial pulse, however, was noted for more than a week before death, and this was observed to be due to an obstruction which could be traced to the bifurcation of the brachial artery. The brachial artery itself pulsated. Sepsis, due to some pus-forming organism, seemed to be a plausible explanation of all the symptoms, but its mode of entrance into the system and its chief habitat remained a mystery to the end.

Post mortem the patient was shown to have had small multiple abscesses in the brain, a large embolus in the left brachial artery (Fig. 5) which had been recognized in life, and a large recent infarct in one kidney—all caused evidently by an immense vegetating mass which was found on the inner wall of the aorta at its arch. A drawing of this vegetating mass is given here. (Fig. 6.) It may be observed from this drawing that this mass is almost directly in front of the orifices of the great arteries of the head and arms—*i. e.*, the innominate, the carotid, and the subclavian. From it, as from a centre, small emboli had been carried to the brain, where they became each the focus of a small abscess, and to other organs and parts, especially into the brachial artery and to the kidney.

EXTERNAL PILES AND THEIR RELATION TO THE EXTERNAL HEMORRHOIDAL VEINS.

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THE swellings and vascular tumors occurring at the lower extremity of the alimentary canal, known as hemorrhoids or piles, are very naturally classified, according to their position, as either external or internal. This classification is based not only on the fact that one group is in view and covered with skin, while the other is concealed within the bowel and covered with mucous membrane, but also on account of their relations to the external and internal hemorrhoidal veins respectively.

As the essential element in a large majority of hemorrhoidal swellings is a varicose condition of the hemorrhoidal veins, the study of this disease cannot lead to any satisfactory results unless based upon a comprehensive and accurate knowledge of the arrangement of these veins. The hemorrhoidal veins comprise the internal or superior hemorrhoidal,